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CORRESPONDENCE

Letters to the Editor

Clinical Value of Myocardial Contrast Delayed Enhancement With Multidetector Computed Tomography

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We read with great interest the article by Sato et al. (1) that evaluated the prognostic value of myocardial contrast delayed enhancement with 64-slice multidetector computed tomography (MDCT) after acute myocardial infarction. They should be commended for conducting the study elegantly. However, a few interesting points arise from the analysis that we believe may be pertinent and should be answered.

Because MDCT can detect peri-infarct tissue heterogeneity that can trigger ventricular arrhythmias (2), which are a common cause of morbidity and mortality in patients who have had a myocardial infarction. Did the authors make an attempt to evaluate and correlate peri-infarct heterogeneity with prognosis? Also, automated implantable cardioverter-defibrillators (AICDs) are known to improve the prognosis in a subset of patients who have had a myocardial infarction (3), so it would be interesting to know the AICD distribution among various tertiles and among event groups and whether the authors adjusted its distribution as a possible confounding factor.

Were patients with high levels of biomarkers that are known to be associated with poor prognosis and are highly correlated with delayed enhancement size (creatine kinase-myocardial band, troponin, poor left ventricular ejection fraction) treated more aggressively, or was any attempt made to standardize the therapy after hospital discharge?

Because the number of segments with transmural infarct (on the 17-segment model) is a strong and independent predictor of prognosis (4), it would be interesting to know if authors made an attempt to determine which is the better prognostic indicator: the number of segments involved or infarct size (e.g., multiple segments with subendocardial infarct versus few segments with transmural infarct).

Did the authors make any attempt to study the significance of calcium deposits in acute infarct detected on MDCT and its impact on prognosis?

MDCT is an excellent alternative in situations where magnetic resonance imaging (MRI) is contraindicated and attributing to its improving resolution and reduced partial volume effect that results in a more accurate assessment of area amenable to revascularization, which might replace MRI in the future. However, the absence of a universally acceptable protocol for delayed imaging is a critical road block due to the time dependent nature of the contrast uptake. Another limitation is hyperenhancement of both acute and chronic infarct, which limit the role of delayed enhancement on multidetector computed tomography in patients with a history of myocardial infarction.

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Reply

We thank Drs. Sharma and Chatterjee for their comments in regards to our recently published paper (1). A previous report demonstrated that multidetector computed tomography could detect peri-infarct tissue heterogeneity 6 months after myocardial infarction (MI) that could trigger ventricular arrhythmias (2). However, we could not detect peri-infarct tissue heterogeneity and calcium deposits of infarcts immediately after primary percutaneous coronary intervention. Acute MI is associated with myocardial edema during the acute phase (3), and therefore, this also may influence the extent of myocardial contrast delayed enhancement. We agree that automatic implantable cardioverter-defibrillators (AICDs) are known to improve the prognosis in a subset of patients who have had an MI, but only a few patients received AICD therapy in our study. There is a low incidence of sudden cardiac death in survivors of MI in Japan. During an average follow-up of 4.1 years, 1.2% of 4,122 consecutive patients with acute MI discharged from the hospital had sudden cardiac death (4). AICDs are implanted only in high-risk patients with cardiac dysfunction (left ventricular ejection fraction <40%), nonsustained ventricular tachycardia, and sustained ventricular tachycardia induced during an electrophysiological study.

The purpose of our study was to evaluate the clinical value of myocardial contrast delayed enhancement with multidetector computed tomography for predicting clinical outcome after acute MI. Therefore, our patients were treated with standard therapy after hospital discharge.

The number of left ventricular segments with transmural delayed enhancement has been shown to be a major factor for the

prediction of prognosis (5). According to univariate analysis in our study, the number of left ventricular segments showing transmural extent was associated significantly with cardiac events. After adjustment for multiple confounders, this parameter lost its predictive power, and myocardial contrast delayed enhancement size was found to be the only significantly independent predictor of cardiac events. The definition of hyperenhancement has not been standardized; thus, there was a limitation because of the use of a global classification, in particular for patients with both transmural and subendocardial hyperenhancement. Specific optimal cutoff values for normal and infarct cores are unknown and are likely dependent on study quality. Further large studies to confirm these findings in clinical examinations will be needed.

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The Predictive Value of Plasma Neutrophil Gelatinase-Associated Lipocalin on Cardiovascular Death and All-Cause Mortality Might Be Mediated by Leukocytosis

We read with interest the paper by Daniels et al. (1), who concluded that higher levels of plasma neutrophil gelatinase-associated lipocalin (NGAL) are associated independently with an increased risk of cardiovascular death and all-cause mortality in a cohort of older community-dwelling adults. The lack of correlation with renal function led the authors to conclude that plasma NGAL provides prognostic information that is independent of glomerular filtration rate, and the modest correlation with C reactive protein also underlines that NGAL would reflect a pathophysiological process distinct from inflammation. We (2), as well as others (3), have shown independently that serum, but not urinary, NGAL levels are influenced dramatically by leukocytosis, especially by a high neutrophil count. The paper by Daniels et al. (1) lacks data regarding leukocyte or neutrophil counts, and therefore, it cannot be excluded that the significant role of plasma NGAL in predicting mortality and cardiovascular outcomes observed in this study might have been mediated largely by these untested parameters, because leukocytosis is per se a significant predictor of noninfective mortality and morbidity, particularly resulting from cardiovascular or cerebrovascular causes (4), thereby biasing their results.

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Reply

We thank Drs. Lippi and Cervellin for their interest in our study (1). Although leukocyte count was not measured at the 1992 through 1996 Rancho Bernardo study visit, 802 (58%) of the 1,393 individuals in our study did undergo leukocyte count measurement in a previous visit approximately 4 years earlier. In these individuals, the correlation between potentially log (neutrophil gelatinase-associated lipocalin) and leukocyte count was only 0.14. We repeated the multivariable Cox proportional hazard models with leukocyte count included as a covariate, and the results were essentially unchanged, with only a very slight attenuation of the risk estimates. Neutrophil gelatinase-associated lipocalin was still a strong, independent predictor of cardiovascular disease death and of the combined cardiovascular end point in fully adjusted models that included leukocyte count. In addition, the association with all-cause mortality remained significant in models adjusted for age, sex, and leukocyte count, with borderline significance after adjusting further for other risk factors. Thus, neutrophil gelatinase-associated lipocalin seems to be an independent predictor of cardio-